ACUTE MASTOIDITIS

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Anatomy of the Mastoid Process.

The mastoid process is shaped like a cone with the base uppermost.

Above, it is separated from the temporal fossa by the supra-mastoid crest which is continued forwards over the external auditory meatus as the posterior root of the zygoma.

Posteriorly, the mastoid process articulates with the occipital bone and above this with the mastoid angle of the parietal bone.

Anteriorly, the process forms the posterior wall of the external auditory meatus.

The tip is free and directed downwards. On its outer surface the tendinous fibres of the sterno-mastoid muscle are attached and on its medial side is a groove which lodges the posterior belly of the digastric muscle.

Immediately above and behind the postero-superior curve of the external auditory meatus there is a triangular cribiform area, which marks the site of the underlying mastoid antrum.

On or near the masto-occipital suture is an opening which transmits the mastoid emissary vein: the vein links up the transverse sinus with the occipital vein.

On its cranial surface the mastoid process has a broad shallow groove which lodges the transverse sinus and its continuation the sigmoid sinus.

On section, two main types of mastoid process are differentiated, cellular and non-cellular.

I. Cellular type of mastoid process. The mastoid antrum is situated in the upper and anterior part of the process. Continuous with the antrum there is a honey-comb network of cells which spreads through the mastoid process. The cells are very variable both in their size and in their distribution. Frequently they extend throughout the process with larger cells at the tip and behind the groove of the sigmoid sinus.

Cells may extend backwards beyond the masto-occipital suture into the occipital bone. They may invade the supra-mastoid crest and continue upwards into the squamous temporal, or forwards along the posterior root of the zygoma roofing over the external auditory meatus and reaching the zygomatic process itself. Cells may surround the external auditory meatus and the Eustachian tube. They may invade the petrous portion of the temporal bone, surrounding the bony capsule which encloses the internal ear and reaching as far as the tip of the petrous bone where it articulates with the sphenoid.

The mastoid cells are separated from the surface and from the dura mater of the middle and posterior fossæ by thin plates of bone.

All cells are lined with mucous membrane which is directly continuous with the mucous membrane of the mastoid antrum and of the middle ear.

The distribution of the mastoid cells is of the greatest clinical importance as it governs the extent and direction of an inflammatory process spreading from the middle ear.

2. Non-cellular type of mastoid process. On section the mastoid process is seen to consist of ivory dense bone or of diploëtic bone. The only cellular spaces are the mastoid antrum and one or two small air cells immediately surrounding the antrum.

This type of mastoid process is probably due to a failure of the normal process of pneumatisation and is not the result of a previous inflammatory process in a cellular mastoid.

Both mastoid processes are usually roughly symmetrical. Both are usually cellular or both non-cellular—in the cellular types there is commonly a fairly close resemblance in the distribution of the cells on both sides, but exceptions to this rule occur and one mastoid process may be cellular while the other is non-cellular.

The mastoid process in infancy. In infants the mastoid process is not developed but the mastoid antrum is present at birth and is as large relatively as the antrum in the adult. In infancy the antrum lies superficially, separated from the subcutaneous tissues only by a thin plate of bone.

Cells begin to appear soon after birth and by the time the child is four to five years old the process is well developed.

One important result of the absence of a mastoid process in the young infant is that the facial nerve, as it emerges from the stylo-mastoid foramen, is unprotected by any overlying bone and may be injured by a skin incision extending into the neck.

The middle ear cleft. The mastoid antrum communicates with the upper part of the middle ear cavity through a short wide passage—the aditus. In its turn the middle ear cavity communicates with the naso-pharynx through the Eustachian tube. This series of cavities and communicating passages may be called the middle ear cleft. The cleft from the naso-pharyngeal orifice of the Eustachian tube to the most distal mastoid air cell is lined throughout by a continuous mucous membrane.

An inflammatory process of the naso-pharynx passes to the middle ear cavity by way of the Eustachian tube. When a suppurative otitis media has developed, the close proximity of the mastoid antrum and air cells and the continuity of the mucous membrane ensure some degree of inflammation in these cavities. This is most clearly evident in children or in adults with extensive pneumatisation of the mastoid process, and is shown by tenderness on pressure over the mastoid process. The tenderness is most marked on pressure applied immediately over the mastoid antrum and lessens progressively over the mastoid process. The tenderness is greatest before the tympanic membrane has ruptured and becomes progressively less as drainage through a perforation becomes established, but it may take several days to disappear completely. The tenderness indicates an early stage of mastoiditis but does not call for a mastoid operation, provided that it continues to diminish, there is no pain and the general condition improves progressively.

If an X-ray of the mastoid process is taken at this stage the mastoid cells will appear hazy, owing to thickening of their mucous membrane lining and, perhaps, to the presence of exudate.

Course of a mastoiditis after rupture of the tympanic membrane. There are three possibilities :

1. *Most commonly a cure results.* The perforation in the membrane is sufficient to ensure drainage. The patient's resistance overcomes the virulence of the infecting organism, all acute signs disappear, the discharge becomes progressively less and ultimately ceases. The perforation in the tympanic membrane heals.

2. *A chronic or latent mastoiditis develops.* The perforation in the membrane gives sufficient drainage to relieve the patient of all acute signs of mastoid infection. The mastoid tenderness slowly lessens and ultimately disappears entirely: convalescence is protracted, evening temperature remains high for some days after the appearance of the discharge, and pain persists, but gradually lessens, until finally the only indication of mastoid infection is a persistent discharge from the ear. This discharge is purulent in character and is often profuse. It may persist without symptoms for months, but there is a tendency for acute exacerbations to occur. An exacerbation is often heralded by a disappearance or lessening of the discharge and this is followed by a renewal of pain, mastoid tenderness and pyrexia over a few days.

An X-ray of the mastoid processes taken at any time during the course of the discharge will show that the mastoid cells on the affected side are cloudy and blurred. A chronic mastoiditis of this nature calls for drainage of the infected mastoid process. Operation is best undertaken during a quiescent period.

3. *An acute mastoiditis develops.* The drainage supplied by the perforation in the membrane or the resistance of the patient is not sufficient to overcome the infection. The oedematous mucous membrane of the antrum and mastoid cells becomes ulcerated. The underlying bone is deprived of its blood supply and becomes infected in its turn, with destruction of the bony cell walls.

The acute symptoms and signs of the otitis media become intensified and it is now necessary to drain the infected mastoid process. The symptoms and signs of this stage vary enormously, according to the type of mastoid process, and they will be discussed as they occur (*a*) in a cellular and (*b*) in a non-cellular mastoid process.

Signs of Acute Mastoiditis in a Cellular Mastoid.

Pain. The severe pain of an acute otitis media is relieved by the perforation of the tympanic membrane. As an acute mastoiditis develops, pain returns but is rarely so severe as during the initial stages. It is throbbing or boring in character, deeply-seated and is situated rather over the mastoid process than in the ear itself. It is usually worst at night so that sleep is interrupted. The pain is often greatly relieved by the application of warm dressings or by the administration of aspirin.

The recurrence of pain is the usual but not invariable accompaniment of an acute mastoiditis. Infection with a pneumococcus type III organism (*streptococcus mucosus*) may run a painless course.

Temperature. During the onset of the infection, in the stage of acute nasopharyngitis and otitis media, the temperature is persistently high. After drainage of the middle ear has been established through a perforation in the tympanic membrane the temperature tends to drop steadily to normal, in the course of two or three days. As an acute mastoiditis develops pyrexia recurs but rarely to a level so high as during the initial stages. This second onset of temperature is

rarely a persistent temperature but shows a daily swing, low in the morning and progressively higher in the evening. It is of the utmost importance, therefore, during the few days which follow the onset of the discharge to keep a regular daily chart showing morning and evening temperature, and great significance must be attached to it, particularly in the absence of other complications such as cervical adenitis.

Tenderness. There are no more misleading signs in acute mastoiditis than the presence or absence of tenderness.

Its presence depends on the degree of pneumatization of the mastoid process and the degree of thickness of the bone forming the cortex of the mastoid process and separating the infected cells from the surface. Where cells are numerous and large and the cortical bone is thin, tenderness is an early sign. When cells are few and the cortical bone thick, tenderness will be late in its appearance or may never be present.

At first tenderness in a well pneumatized process is most marked at three points—over the mastoid antrum, over the tip and over the postero-inferior border of the process. As the infection develops tenderness becomes generalised over the mastoid process.

If operation is delayed tenderness may again become more localised, either over a group of particularly large cells near the surface, or at a point where perforation of the cortical bone is impending.

True mastoid tenderness is a deep-seated tenderness elicited only by firm pressure on the bone. A superficial tenderness, due to an infection of small lymph glands over the mastoid process, must not be mistaken for true mastoid tenderness.

Mastoid tenderness must not be confused with the tenderness caused by a furuncle of the external meatus. The latter is elicited by displacement of the auricle and is commonly most severe on pressure over the tragus.

Oedema of the periosteum and of the cellular tissues overlying the mastoid process. In a very cellular process with thin cortical bone this oedema may be an early sign, present for many days before perforation of the cortex occurs and a subperiosteal abscess is formed. It is detected by gentle palpation and comparison with the healthy mastoid. The skin over the mastoid process may be slightly reddened. If the mastoid processes are viewed from behind, the auricle on the affected side will appear slightly more prominent.

Formation of a subperiosteal abscess. A subperiosteal abscess is formed by perforation of the cortical bone from an infection of a closely underlying cell. It is a very frequent and comparatively early complication in the very young. The formation of the abscess is shown by a great increase in the periosteal oedema and an obvious swelling over the mastoid process which displaces the auricle. Usually the perforation occurs in the bone immediately overlying the mastoid antrum and the resultant swelling displaces the auricle downwards and forwards.

This swelling is not pathognomonic of acute mastoiditis as it develops similarly in many cases of furuncle of the external meatus. In furunculosis there is a localised swelling in the outer half of the external auditory meatus and the tympanic membrane is intact and not bulging.

Sometimes, unfortunately, furunculosis coincides with an otitis media. The differential diagnosis of the mastoid swelling may then become very difficult. Help may be obtained by X-rays, which will show the disintegration of cell walls in acute mastoiditis, but if this is not available exploration may be required, and should be undertaken in cases of real difficulty.

If the condition is not relieved by operation the skin over the abscess may ultimately redden and break down with the formation of a sinus through which bare bone can be probed.

Rarer sites for the formation of a subperiosteal abscess.

(a) Below the tip of the mastoid process under cover of the attached sternomastoid muscle.

(b) Above the auricle over the zygomatic process or squamous temporal.

(c) Along the postero-inferior border of the mastoid process.

These sites are determined by the presence of large infected cells, either at the tip of the process or extending forwards along the posterior root of the zygoma, or upwards into the squamous temporal, or, finally, behind the groove for the sigmoid sinus.

(a) This type is called a Bezold's mastoiditis. The perforation takes place through the medial surface of the mastoid process and the abscess extends along the posterior belly of the digastric or under cover of the sternomastoid muscle and tends to track down the neck in the line of the great vessels. There is usually great tenderness on pressure over the tip of the mastoid.

This swelling must not be confused with the swelling which is the result of an inflammation in the uppermost glands of the anterior triangle of the neck, secondary to the initial nasopharyngitis and otitis media. The swelling due to an adenitis is situated below the tip of the mastoid process, which can be separated from it by careful palpation and which is not tender on deep pressure. The swelling of a Bezold's mastoiditis is directly continuous with the tip of the mastoid process.

(b) Zygomatic mastoiditis is the term applied to the formation of a subperiosteal abscess over the zygoma or in the temporal fossa. The swelling is visible above and in front of the auricle and invades the upper and lower eyelids on the same side. In the cellular tissues of the eyelids the oedema is most evident and will often be the first indication of this type of mastoiditis.

(c) A subperiosteal abscess may form on the postero-inferior aspect of the mastoid process, due to an escape of pus from cells which extend backwards and downwards beyond the curve of the sigmoid sinus. The mastoid emissary vein pierces the cortex at this site and pus may escape through the perforation which transmits the vein, either from infected cells in the vicinity or from a perisinous abscess. There may be an accompanying thrombosis of the vein or of the sinus, or of both.

Otoscopic Examination.

The tympanic membrane is congested, oedematous and bulging in its postero-superior quadrant. The bulge may take the form of a nipple-shaped projection, pointing downwards and forwards, perforated at the tip. The discharge from the perforation is purulent, pulsating and profuse, so that it wells into the external

meatus in quantity so great that it obviously comes from a wider area than the small tympanic cavity itself. Co-extensive with the oedema of the membrane there is a sagging of the postero-superior meatal wall.

A profuse discharge is also seen in severe Eustachian infections but it is more muco-purulent in character, stringy and sticky, and if carefully wiped away, it can be seen welling up from an anterior perforation opposite the mouth of the Eustachian tube.

It is essential in difficult cases to take great care with the otoscopic examination to differentiate the discharge which is coming from an infected mastoid process from the discharge of a persistent Eustachian infection. The essential points are:

1. The sagging and oedema of the postero-superior deep meatal wall in acute mastoiditis.
2. The site of the bulge in the tympanic membrane—a bulge from above and behind, downwards and forwards in acute mastoiditis.
3. The nature rather than the quantity of the discharge. Thick, creamy and purulent in acute mastoiditis—not muco-purulent.

Constitutional symptoms. These are prominent in acute mastoiditis. Head-ache, loss of appetite and, above all, sleep disturbed by bouts of pain.

X-ray diagnosis. The diagnosis should be made by physical symptoms and signs and not by X-ray, except in a few difficult cases. X-rays can be most misleading because in all cases of acute otitis media, even when there is not sufficient infection in the mastoid process to warrant operation, the mastoid cells will appear cloudy. A true mastoiditis will be shown by softening and breaking down of the cell walls with abscess formation.

In spite of these difficulties, an X-ray of the mastoid process should be taken before operation as it gives an accurate picture of cell distribution and of the spread of the disease and enables the surgeon to plan the scope of his operation.

Acute Mastoiditis in a Non-cellular Mastoid.

If the mastoid process is not cellular but of the diploetic type, an acute mastoiditis takes the form of a spreading osteomyelitis which involves the plates of bone covering dura mater before it reaches the surface through a dense cortex. The disease in this case is therefore particularly dangerous and is apt to lead to intra-cranial complications.

If the mastoid process is sclerosed, an acute mastoiditis is unlikely to develop—it is in this type that a chronic suppurative otitis media often follows.

Acute mastoiditis in a diploetic mastoid is a particularly dangerous type of mastoiditis and all the more so because diagnosis is difficult and operation tends to be delayed.

1. *The usual physical signs of mastoiditis are absent.* Owing to the dense cortex there is no mastoid tenderness. There is no oedema of the periosteum, no displacement of the auricle. A subperiosteal abscess will not form till long after the infection has reached the dura mater. It is important then that the few signs that persist should not be missed.

2. *On otoscopic examination* there is sagging of the deep meatal wall because the mastoid antrum lies in close proximity beneath it. There is bulging of the tympanic membrane with the nipple shaped type of perforation, as in a cellular mastoid. There is a profuse pulsating purulent discharge. Too much stress can scarcely be laid on these very vital signs.

3. *The general condition of the patient.* There is persistent pain in the ear and deep seated headache after the discharge has been fully established. The patient shows signs of marked toxæmia, with loss of appetite, constipation and sleeplessness. The temperature remains high and tends to be of the swinging septic variety.

To sum up, in this most dangerous type of acute mastoiditis there is no tenderness, no mastoid oedema, no displacement of the auricle. The diagnosis must therefore be made by otoscopic examination and consideration of the patient's general condition.

Atypical Forms of Acute Mastoiditis.

1. **Petrositis.**—Acute mastoiditis complicated by the spread of infection into the petrous part of the temporal bone. This is most likely to occur when there is an extension of cells into the petrous bone. These cells surround the dense bony capsule of the labyrinth and extend to the tip of the petrous.

The signs and symptoms usually appear after an acute mastoiditis has been fully established and often during the convalescence following a mastoid operation. They may, however, appear at the beginning of an acute mastoiditis.

The most prominent symptom is neuralgic pain over the temporal region on the same side, radiating downwards over the cheek and into the teeth, or a deep-seated boring frontal headache, round and behind the eyeball.

This pain is most likely caused by irritation of the Gasserian ganglion of the fifth nerve as it lies on the apex of the petrous bone.

The sixth nerve also crosses the tip of the petrous bone and may be involved. As a result there is paralysis of the external rectus muscle on the same side with a resultant squint and diplopia.

The infection round the labyrinth may cause symptoms of vertigo with spontaneous nystagmus.

There is a continued pyrexia of the swinging septic type, and also persistent, copious, purulent discharge from the meatus.

A suppurative process of the petrous bone untreated is exceedingly dangerous, as the infection readily extends into the cranial cavity with a resultant meningitis either localised and serous or, more fatally, spreading and suppurative.

Alternatively, the pus may rupture through the floor of the tip of the petrous bone and cause a lateral pharyngeal abscess. In this case there is bulging inwards of the tonsil and of the whole lateral pharyngeal wall with trismus.

It is most important to recognise that the symptoms of post-operative trigeminal pain and paralysis of the external rectus may arise without a suppurative process in the petrous bone. The symptoms are then most probably toxic in origin and operation on the petrous bone should be avoided. In cases of doubt an X-ray view of the petrous bone itself is invaluable for diagnosis.

2. **Mastoiditis due to infection with the streptococcus mucosus (pneumococcus type III).** Infection by this organism has a sinister reputation because it leads to a widespread destruction of bone with few symptoms beyond a purulent ear discharge.

There is little or no pain or tenderness and an intracranial complication may be the first serious sign of trouble. The infection is commoner in the old and those suffering from debilitating diseases, such as diabetes.

3. **The acute mastoiditis of measles.** In the young child an acute otitis media is often thought to be painless. This in many cases is probably due to the failure to interpret the signs of distress in a toxic child during the short interval which elapses between the onset of the infection and the perforation of the membrane with the appearance of discharge. The infection is almost always due to an hæmolytic streptococcus and, in a child of low resistance, there is a rapid destruction of the tympanic membrane with ulceration of the mucosa of the tympanum, antrum and mastoid cells and spreading osteitis.

As a result, complications are relatively frequent or a chronic suppurative otitis media is established which does not yield to treatment.

4. **Acute mastoiditis with a normal tympanic membrane.** It is not uncommon to get a fully developed mastoiditis with an *intact* tympanic membrane. The middle ear as well as the mastoid process is infected and contains pus. The tympanic membrane is injected and bulging. A paracentesis of the membrane should be done at the same time as the mastoid operation.

It is very much less common to get an acute mastoiditis with a *normal* tympanic membrane. It is theoretically possible for this to occur when the infection has reached the mastoid process by way of the blood stream, instead of by the usual route through the middle ear. It is much more likely that there has been a transient otitis media which has resolved without perforation of the membrane, leaving behind it a localised abscess in a group of mastoid cells. A history is then obtained of a preceding attack of pain in the ear, followed—after an interval of some days or even weeks—by tenderness in a strictly localised area over the mastoid process or even by the formation of a localised subperiosteal abscess. Operation will reveal pus in an outlying group of mastoid cells—the remainder of the mastoid process and the middle ear being uninfected.

It is difficult to believe in a generalised infection of the mastoid antrum and air cells without some changes in the tympanic membrane being visible to a careful observer.

5. **Acute osteomyelitis of temporal bone.** This is a rare but very serious type of infection with a high mortality.

The infection may spread to the temporal bone directly from neighbouring bones of the cranial vault. The infection may reach the temporal bone by way of the blood stream from a distant focus. The osteomyelitis may be due to a direct spread from a mastoid infection. There is œdema over the mastoid and in the temporal fossa, great pain, high fever and prostration. Intra-cranial complications and metastatic abscesses are common sequelæ.

Indications for Operation.

There are few physical signs which are absolute indications for operation. Persistent œdema over the mastoid process, formation of a subperiosteal abscess,

or persistent sagging of the postero-superior deep meatal wall are the principal definite indications.

Failing these, careful consideration has to be given to a combination of many minor points.

The general condition of the patient should steadily improve after the appearance of the discharge. Appetite should return and sleep be undisturbed by attacks of pain. The temperature should gradually return to normal and not tend to swing upwards in the evening. Tenderness should grow progressively less. The discharge may remain copious but should not become purulent.

While it is a good thing to operate when the infection has become localised as much as possible, it is exceedingly dangerous to wait for localisation to take place in face of a progressive deterioration in the general condition of the patient.

Scope of the operation. The operation aims at opening and giving free drainage to the infected mastoid antrum and to all the infected mastoid cells. This will entail a careful search for cells. Failure to find and drain infected cells may have serious consequences. An X-ray will afford an excellent guide, but careful operative technique is better still.

It is not necessary to remove the plates of bone covering the dura mater and lateral sinus as a routine, but it is better to expose dura mater unnecessarily than to leave an extra-dural collection of pus undrained.

Complications. 1. *Extradural abscess* usually gives rise to no symptoms and is found at operation by a careful following up of infected cells. It requires no treatment beyond free drainage.

2. *Thrombosis of the lateral sinus* will be immediately suspected if the patient has abrupt rises of temperature to 103°F. or higher, accompanied by a rigor and followed by sweating and an abrupt fall of temperature.

3. *Abscess of the temporal lobe or abscess of the cerebellum* will give rise to signs of increased intra-cranial tension with localising signs varying with the site of the abscess and the degree of surrounding encephalitis.

4. *Infective meningitis* will be diagnosed by severe headache, neck stiffness and head retraction, high temperature and changes in the cerebro-spinal fluid, obtained by lumbar puncture.

5. *Labyrinthitis* will give rise to profound deafness, severe vertigo and the onset of spontaneous nystagmus.